

arachnoid alcohol blocks. All had advanced malignant disease and intractable pain. Pain relief was classified as good in 58 per cent, fair in 28 per cent, poor in 10 per cent, and no pain relief in 4 per cent. There were no deaths. There were four complications (3.7 per cent) in the series: Two patients had bladder dysfunction requiring catheter drainage for 72 hours; one patient had left deltoid muscle group paralysis after a right C<sub>5-6</sub> subarachnoid block; the remaining patient had motor weakness in the left hip flexors and quadriceps with slight residual weakness after seven days.

Based upon these observations, we believe that chemical rhizotomy should be considered for relief of intractable pain secondary to malignant disease before one undertakes more hazardous surgical procedures.

**Electrocardiographic Studies of the Tracheo-Cardiac Reflex During Light Analgesia with Nitrous Oxide and Relaxants.** MORRIS L. HELLER, M.D., AND T. RICHARD WATSON, JR., M.D., Departments of Anesthesiology and Surgery, Dartmouth Medical School, Hanover, New Hampshire.

CARDIAC arrhythmias occur during laryngoscopy and intubation. Numerous reports state that light anesthesia contributes to their development [ANESTHESIOLOGY 11: 224, 1950]. It is also intimated that one of the factors often responsible for cardiac arrest is insufficient anesthesia in the presence of strong sensory stimulation; this would include intubation [ANESTHESIOLOGY 16: 177, 1955]. To prevent these abnormal rhythms, it has been suggested that there is greater safety in the deeper anesthetic levels.

This report is concerned with disturbances of cardiac rhythm and their mechanism during laryngoscopy and intubation of the tracheas of 106 patients under light nitrous oxide-relaxant anesthesia. No thiopental, cyclopropane, or ether was added, and topical anesthesia was omitted. Electroencephalographic studies made by us have shown that during nitrous oxide analgesia the brain-wave does not show significant variation from the control tracing.

Of the 106 patients studied, 71 showed no change after laryngoscopy; 14 showed an increased heart rate; 21 demonstrated a tendency toward bradycardia, but the rate became more rapid after intubation; two patients developed a nodal rhythm; and one presented a single nodal premature beat.

Following tracheal intubation, there was no change in 24 patients; 72 showed an increased heart rate; 10 developed a slower rate with return of a faster rhythm within 30 seconds in eight; there were two instances of nodal rhythm; and one patient showed solitary premature auricular beats. Sinus arrhythmia was noted occasionally.

The control of the heart through autonomic balance is vividly portrayed during light nitrous oxide analgesia. The majority of the patients in this study responded with mild sympathetic stimulation manifested by increased cardiac rate. Sympathetic stimulation evidenced by frequent ventricular extrasystoles, bifocal or multifocal ventricular tachycardia did not occur with light anesthesia. This has been observed with the more potent anesthetic agents, where there is good evidence for myocardial depression associated at times with hypoxia and hypercapnia.

A temporary increase in vagal tone was observed in a small number of patients during analgesia. Four patients (3.8 per cent) showed a displacement of the pacemaker and nodal rhythm. In every instance of nodal rhythm normal or faster rate returned within one-half minute. Vagal disturbances occurred abruptly; whereas sympathetic cardiovascular responses developed more slowly over a period of several seconds.

We do not believe it necessary to depress parasympathetic reflexes by utilizing deeper levels of anesthesia or with injection of large doses of atropine or by topical spraying. There is adequate protection through the sympathoadrenal stimulation which is active under light anesthesia. This sympathetic response is a salutary one, and is an important safeguard in maintaining cardiovascular homeostasis during anesthesia and surgery. (Supported in part by grants from the New Hampshire Heart Association and the National Heart Institute (H-3154) of the National Institutes of Health, U. S. Public Health Service.)